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Smoking or poor oral hygiene do not predispose to peritonsillar abscesses via changes in oral flora

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Informed consent: All patients and healthy volunteers provided written informed consent to participate in the original study and for use of their samples also in future studies.

Abstract

Aim: The purpose of this prospective study was to determine if there is a difference in number and distribution of salivary bacteria between patients with tonsillar infection and healthy volunteers.

Background: The aetiology of peritonsillar abscess (PTA) is unclear. Smoking, periodontal disease, and infection of minor salivary glands have been suggested as predisposing factors for PTA.

Material and Methods: Patients with acute tonsillitis (AT) (n=54), peritonsillitis (PT) (n=36), PTA (n=58), and healthy volunteers (n=52) were prospectively recruited and evaluated. Saliva bacteria were analysed with flow cytometry. Patients and their treating physicians completed a questionnaire about patients' current disease, smoking habits, alcohol consumption, and oral health.

Results: There were no differences in the total number of saliva bacteria between patients with acute throat infection and healthy volunteers (P=0.104) or between AT, PT, and PTA patients (P=0.273). Smoking habits, alcohol consumption, oral hygiene, or prior antibiotics had no effect on total amount of salivary bacteria in patients with acute throat infection.

Conclusion: The effects of smoking on salivary bacteria do not seem to be the mechanism that promotes development of PTA in smokers.

Significance: This study challenges the role of poor oral hygiene as an aetiological factor for PTA.

Abbreviations: Peritonsillar abscess (PTA), acute tonsillitis (AT), Peritonsillitis (PT), recurrent tonsillitis (CT), Group A streptococcus (GAS), *Fusobacterium nucleatum* (FN), *Streptococcus anginosus* group (SAG), *Porphyromonas gingivalis* (POGI), phosphate-buffered saliva (PBS), flow cytomety (FCM)

Key words: salivary bacteria, tonsillar infection, flow cytometry

Introduction

Peritonsillar abscess (PTA) is the most common deep otorhinolaryngological (ORL) infection and is traditionally regarded as a purulent complication of acute tonsillitis (AT). However, no strong correlation between the incidence of AT and PTA has been shown. Other factors such as poor oral hygiene, smoking, or infection of small salivary glands have recently been proposed to enhance the development of PTA. [1,2]. Bacterial causes of PTA have been widely investigated. Although there is some consistency between studies, it is unclear why the same bacteria cause aggressive PTA in some patients and uneventful tonsillitis in others.

Like PTAs, oral infections are also typically polymicrobial. Oral infections are caused by the concerted action of commensal bacteria and opportunistic oral pathogens. [3,4]. Some key pathogens have been identified in certain common oral diseases, such as periodontitis and dental caries. These include *Porphyromonas gingivalis* (POGI) in chronic periodontitis and *Streptococcus mutans* in the development of dental caries. [4,3]. Periodontitis is a common infection-induced inflammatory disease of the gingiva and the connective tissue surrounding the tooth. If untreated, periodontitis leads to alveolar bone destruction. PTA patients have periodontal disease considerably more often than patients with recurrent tonsillitis (CT). [2]. Certain bacterial species have been identified in both PTA and periodontitis or in the tonsillar area [1]. Although the causality is unclear, PTA and periodontal diseases are speculated to be of multifactorial origin; instead of a certain pathogen, there may be a synergy of certain bacteria or other factors that influence or predispose to both of these diseases.

Smoking promotes the development of periodontal disease and is also associated with increased risk of PTA [5]. The specific mechanism by which smoking promotes these diseases is unclear. Torre et al. demonstrated histological changes in both lymphoid and non-lymphoid compartments of smokers' tonsils. Changes in cell-to-cell interactions could be one mechanism of how smoking induces PTA. [6]. Smoking alters the composition of subgingival bacterial flora in the development of periodontal disease and increases the depth of periodontal pockets [7,5].

The purpose of this study was to clarify the role of possible changes in oral bacteria in the development of PTA by comparing salivary bacterial composition between healthy volunteers and patients with AT, peritonsillitis (PT), and PTA. We also determined if smoking has an effect on salivary bacteria composition.

Patients and methods

The material consisted of the following two prospectively enrolled groups: patients (n=148) and healthy volunteers (n=52). In patient group, patients suffering from throat pain were recruited and evaluated at the emergency department at the Helsinki University Hospital between February and October 2017. On the basis of clinical diagnosis, patients were further divided into three groups, specifically AT (n=54), PT (n=36), and PTA (n=58). Exclusion criteria for patients were age <15 years and known pancreatic disease. Healthy volunteers were students or recruited from the hospital staff between March and April 2018. For healthy volunteers, the exclusion criteria were acute throat pain, acute or chronic tonsillar infection, and age <15 years.

Questionnaire

The infection group completed a questionnaire about their smoking habits, overall health, current disease, prior and current use of antibiotics, smoking habits, alcohol consumption, and throat infections. The treating physician (a resident or a specialist in ORL) completed a questionnaire concerning patients' oral health and tonsillar findings. Oral health was evaluated by inspection (poor/good) in conjunction of the ORL examination. Healthy volunteers completed a questionnaire about recent throat infections, recent antibiotics during the past 2 months, smoking, oral hygiene, and alcohol consumption. Alcohol consumption was regarded as overuse if the patient reported to be under the influence of alcohol at least five times in a month.

Bacterial analysis of salivary samples

Paraffin-stimulated saliva was collected for 2 minutes from all patients and 2.5 ml of the saliva was used for bacterial analysis. Bacterial concentrations were analysed with flow cytometry according to a previously published method [8] with some modifications.

To isolate bacteria, saliva was aliquoted in 1.5 ml-Eppendorf tubes and centrifuged. The supernatant was discarded and pellets were suspended in 200 µl of phosphate-buffered saline (PBS). Thereafter, suspensions were combined and centrifuged. Supernatant was removed and the pellet was suspended in 1000 µl of 4% paraformaldehyde in PBS (w/v) to fix the bacteria. Glass beads were added to the bacterial suspensions and samples were mixed and incubated on a shaker.

To prepare bacterial stocks, samples were centrifuged and the supernatant was removed and pellets were washed twice with PBS. Finally, the bacterial pellet was suspended in 200 µl PBS and an equal volume of 94% ethanol. The samples were stored at -20°C until analysis.

Total bacterial concentration was analysed by flow cytometry (FCM; BD FACSCalibur). To measure the total amount of bacteria, 4 µl of bacterial stock was diluted in 4 ml of PBS and samples were vortexed and sonicated. A total of 300 µl of bacterial suspension was then mixed with 3 µl of Sytox Orange (Molecular Probes, Eugene, Oregon) DNA-stain (Ex/Em 547/570 nm) for FCM analysis to separate bacteria from non-bacterial material.

For bacterial species-specific FCM analysis, 15 µl of bacterial stock sample was hybridized with 16S rRNA-targeted CY5 indocarbocyanin (Ex/Em 646/662 nm Molecular Probes) -labelled oligonucleotide probes against *Porphyromonas gingivalis* [9], *Fusobacterium nucleatum* (FN) [10], *Streptococcus mutans* (MS) [10], narrow Streptococcus probe (NSP; *Streptococcus anginosus* [SAG], *Streptococcus oralis*, and *Streptococcus mitis)* [11] and broad Streptococcus probe (BSP) [12]. Before FCM, 4 µl of Sytox Orange was added to each sample. The sequence match of the probes with the target species was analysed using the Ribosomal Database Project database (http://rdp.cme.msu.edu/html).

Bacterial concentrations were measured using TrueCount® tubes (Beckton Dickinson, San Jose, CA) containing a known number of fluorescent microbeads. A total of 300 µl of each sample was added to a TrueCount® tube and FCM analysis was continued until 2% of the microbeads were detected. Bacterial samples hybridized with Cy5-labelled probes were analysed using the FL4 detector (661/16 nm) and the samples stained with Sytox Orange DNA-stain by FL2 detector (585/42 nm). From each bacterial stock sample two parallel samples were run. The mean, standard deviation, and coefficient of variation of bacterial concentrations was calculated.

Ethical approval

All procedures performed in this study were in accordance with the ethical standards of the institutional research committee (Ethics Committee of Helsinki and Uusimaa Hospital District) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. All participants provided written informed consent before any study-related procedures were performed.

Statistical analysis

Correlations between smoking, alcohol consumption, signs and symptoms of infection, and salivary bacterial samples were analysed. Calculations were performed by NCSS 8 statistical software (Hintze, J. (2012). NCSS 8. NCSS, LLC. Kaysville, Utah, USA. www.ncss.com.). For statistical reasons, the amounts of total salivary bacteria were divided with 10^6. Log transformation was applied to a variable to make the data conform more closely to a normal distribution. Numerical variables were analysed with Mann-Whitney U-test and Kruskal-Wallis one-way ANOVA. Chi-square test was applied to compare nominal variables. *P* values <0.05 were considered statistically significant.

Results

A total of 148 patients and 52 healthy volunteers were included in the study. Patient characteristics and clinical data are presented in Table 1.

There was no difference in the amount of total salivary bacteria between AT, PT, and PTA groups (*P*=0.273) or between patients with acute throat infection and healthy volunteers (*P*=0.104). Distributions between bacteria in AT, PT, PTA and healthy volunteers is shown in Figure 1. Healthy volunteers had a higher amount of POGI than any other patient group (*P*=0.00003) (median amount of bacteria, AT, 0.257 x 10^6/ml; PT, 0.0426 x 10^6/ml; PTA, 0.0523 x 10^6/ml; healthy volunteers, 0.3715 x 10^6/ml).

Smokers had (*P*=0.00074) lower numbers of FN than non-smokers and ex-smokers (median number, smokers, 0.77 x 10^5/ml; non-smokers, 2.1 x 10^5/ml; ex-smokers, 5.41 x 10^5/ml). Ex-smokers had a significantly higher amount of Streptococci than smokers and non-smokers (*P*=0.028) (median number, smokers, 3.2 x 10^6/ml; non-smokers, 4.5 x 10^6/ml; ex-smokers, 14.6 x 10^6/ml). The amount of SAG bacteria (*P*=0.068), POGI (*P*=0.483), or MS (*P*=0.164) did not differ between patients with different smoking habits. The total amount of bacteria was also independent of smoking habits (*P*=0.0866). In subgroup analysis, ex-smokers in the AT group had a higher number of bacteria in total (*P*=0.0428) than smokers or non-smokers. More specifically, ex-smokers had a higher amount of SAG bacteria (*P*=0.049) and FN (*P*=0.0013) than smokers and non-smokers.

Alcohol consumption had no effect on the total amount of salivary bacteria (*P*=0.141). In a subgroup analysis of bacterial findings, the patients who did not consume alcohol had a higher amount of SAG bacteria (*P*=0.046) and POGI (*P*=0.036).

There was no difference in the total number of bacteria between patients with good or poor oral hygiene (*P*=0.621). Distribution of bacteria in patients with good and poor oral hygiene is shown in Figure 2. Comparison of salivary bacteria in AT, PT and PTA patients with different degree of oral hygiene is shown in Table 2. Statistical analysis between the diagnosis groups with different degree of oral hygiene could not be run because the small amount of hybridised samples of patient (AT n=4, PT n=1, PTA n=3). Prior use of antibiotics for current throat infection had no effect on total amount of salivary bacteria (*P*=0.656) or specific bacterial species. Altogether, 49 of 200 saliva samples were not hybridized. Prior use of antibiotics had no effect on hybridization (*P*=0.348).

Discussion

In this study, we observed that smoking habits or oral hygiene have no effect on the number or distribution of salivary bacteria in patients with tonsillar infection. This suggests that poor oral hygiene is not an independent factor promoting the development of PTA. While the role of smoking in PTA is incontrovertible as demonstrated in the literature, the mechanism is still unclear. Our findings indicate that changes in oral bacterial flora is not a causative mechanism.

The role of periodontal disease in the development of PTA has been widely speculated [2,1]. An association between PTA and periodontal disease has been reported. The prevalence of periodontal disease is elevated in PTA patients compared with CT patients. [2]. Although a link between poor oral hygiene and PTA is unclear, the same bacteria that are regarded as the causative agents of

periodontal disease have also been found in pus samples from PTA patients [2,1]. Brook et al. have shown that in the same patients, there are 10 to 1000 fold more bacteria in saliva during AT than after the infection. An increase of FN antibodies after infection was also noted in the same study. [13]. In our study, there was no difference in the number of salivary bacteria between patients with throat infection and healthy volunteers. We did not, however, compare the number of salivary bacteria in the same patients during and after infection.

Smoking is highly associated with increased risk of PTA in both genders. As many as 30% to 60% of patients with PTA report daily smoking, which is a significantly higher number than the smoking rate in the general population (25.5%). [14,15]. In addition, the number of ex-smokers among PTA patients is high (18%) [15]. The effects of smoking on the oral flora are controversial. In some studies, non-smokers and smokers had alterations in oral bacterial flora [16,17]; contrary findings also exist [18]. We identified smokers who had the lowest number of FN and ex-smokers that had the highest number of Streptococci in their saliva. Our findings differ from those of Moon et al., in which smokers had a higher number of *Fusobacterium nucleatum* in subgingival samples. On the other hand, the results are not completely comparable as earlier studies only analysed patients with periodontal disease. [19]. Other mechanisms on how smoking promotes PTA have also been proposed. Torre et al. demonstrated histological alterations in tonsillar lymphoid and non-lymphoid compartments in smokers, which suggests that smoking may alter immune responses and predisposes to PTA. Similar but milder changes were observed in patients with CT. Although recurrent tonsillitis is known to expose to PTA, there are no studies regarding the role of CT [6]. On the other hand, an Iranian group has shown that salivary flow rate is significantly lower in smokers. This could be one explanation for the higher rate of oral diseases among smokers, which could in turn be reflected in the health (or disease) of the tonsillar area. [20]. Our findings challenge the hypothesis that smoking promotes PTA by changing the oral bacterial flora.

We also observed that healthy volunteers had a significantly higher amount of POGI than the AT, PT, and PTA groups. POGI is an important pathogen in the development of periodontal disease [3]. Smoking has not been shown to change the amount of POGI in oral flora [19]. Our finding does not support the hypothesis that periodontal disease causes throat infections, in particular PTA.

Limitations

In our study, oral hygiene was evaluated by a physician, not a dentist, and no standardized evaluation scale or index was used. Statistical comparison between different diagnosis groups and degree of oral hygiene could not run because of the small number of patients with poor oral hygiene in certain diagnosis groups. As none of the healthy volunteers smoked or abused alcohol, these parameters could not be compared with the patient group. The patient groups and healthy volunteers were comparable regarding other variables. Our material is consistent with previous studies concerning patient age, gender, smoking habits, and oral hygiene [2,14,1].

Conclusion

Changes in the number or distribution of salivary bacteria does not appear to be the explanation for the higher rate of PTAs in patients with periodontal disease or smokers. The evidence that periodontal disease causes PTA remains weak and more research is needed to examine the connection between PTA and periodontal disease. While smoking has been repeatedly demonstrated to increase the risk of PTA, the mechanism does not seem to be linked to the distribution or number of salivary bacteria.

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Tables:

Table 1. Characteristics of patients with acute tonsillitis (AT), peritonsillitis (PT), peritonsillar abscess (PTA) and healthy volunteers.

	AT (n=54)	PT (n=36)	PTA (n=58)	Healthy volunteers (n=52)
Age Median Range	 28.5 15-86	 28.5 17-85	 36 16-65	 25.8 19-67
Male, n (%)	21 (38.9)	18 (50.0)	36 (62.1)	19 (36.5)
Smoking, n (%) Yes No Ex-smoker	 22 (40.7) 20 (37.0) 12 (22.2)	 6 (17.1) 21 (60.0) 8 (22.9)	 24 (42.1) 21 (36.8) 12 (21.1)	 0 (0) 52(100) 0 (0)
Alcohol overuse, 5 or more times a month under the influence of alcohol (%)	4 (7.4)	1 (3.4)	5 (8.6)	0 (0)
Prior antibiotics for more than 24 hours, n (%)	9(16.7)	7(20.6)	10 (17.2)	No prior antibiotics
Oral Hygiene, n (%) Good Poor Missing information	 48 (90.6) 5 (9.4) 1	 32 (94.1) 2 (5.9) 2	 51 (91.1) 5 (8.9) 2	 52 (100.0) 0 (0)

AT, acute tonsillitis; PT, peritonsillitis; PTA, peritonsillar abscess

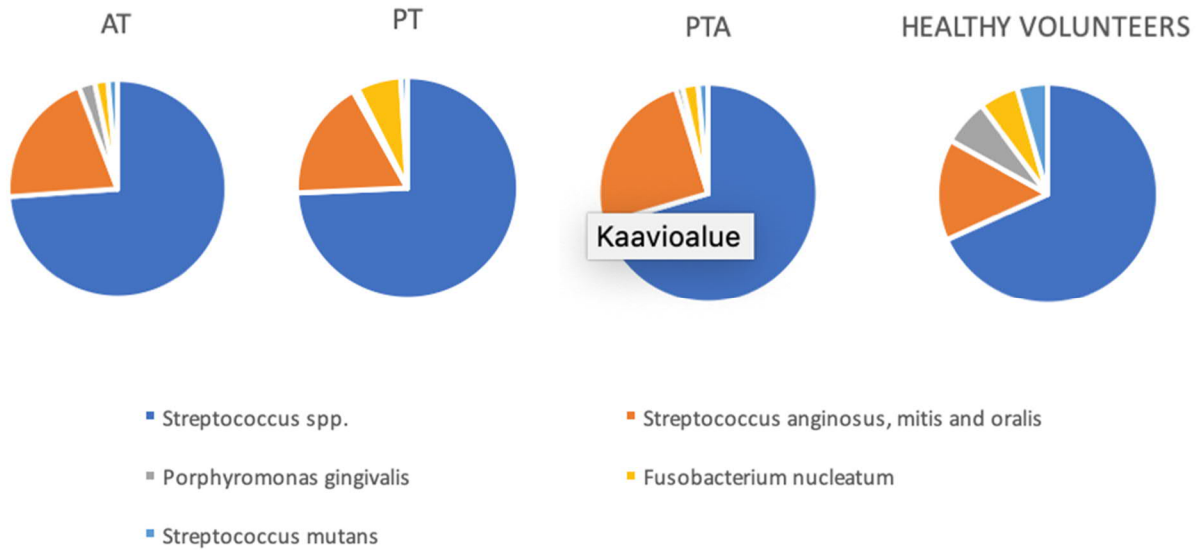
Table 2. Median number of salivary bacteria in AT (n=39), PT (n=24) and PTA (n=22) patients with different degree of oral hygiene (good/poor). Only hybridized salivary samples included.

Median number of salivary bacteria	AT, good oral hygiene (n=35)	AT, poor oral hygiene (n=4)	PT, good oral hygiene (n=23)	PT, poor oral hygiene (n=1)	PTA, good oral hygiene (n=29)	PTA, poor oral hygiene (n=3)
Total number of bacteria (1/ml)	$1{,}04 \times 10^8$	$2{,}42 \times 10^8$	$5{,}06 \times 10^7$	$1{,}18 \times 10^9$	$5{,}71 \times 10^7$	$3{,}33 \times 10^7$
Streptococci group (1/ml)	$8{,}45 \times 10^6$	$1{,}07 \times 10^7$	$3{,}68 \times 10^6$	$4{,}96 \times 10^7$	$4{,}40 \times 10^6$	$2{,}03 \times 10^6$
SAG (1/ml)	$2{,}19 \times 10^6$	$3{,}12 \times 10^6$	$8{,}93 \times 10^5$	$3{,}54 \times 10^7$	$1{,}44 \times 10^6$	$4{,}00 \times 10^5$
POGI (1/ml)	$2{,}43 \times 10^5$	$1{,}57 \times 10^6$	$4{,}26 \times 10^4$	0,00	$5{,}23 \times 10^4$	0,00
FN (1/ml)	$2{,}92 \times 10^5$	$4{,}03 \times 10^4$	$2{,}13 \times 10^5$	$8{,}86 \times 10^6$	$1{,}42 \times 10^5$	$3{,}33 \times 10^4$
S.mutans (1/ml)	$1{,}46 \times 10^5$	$1{,}41 \times 10^6$	$5{,}36 \times 10^4$	0,00	$7{,}59 \times 10^4$	$3{,}33 \times 10^4$

Acute tonsillitis (AT), peritonsillitis (PT), peritonsillar abscess (PTA), *Streptococcus anginosus* group (SAG), *Porphyromonas gingivalis* (POGI), F*usobacterium nucleatum* (FN), *Streptococcus mutans* (*S.mutans*)

Figures:

Figure 1. Distribution of salivary bacteria in AT, PT, and PTA patients and healthy volunteers



AT, acute tonsillitis; PT, peritonsillitis; PTA, peritonsillar abscess

Figure 2. Distribution of salivary bacteria in patients according to oral hygiene

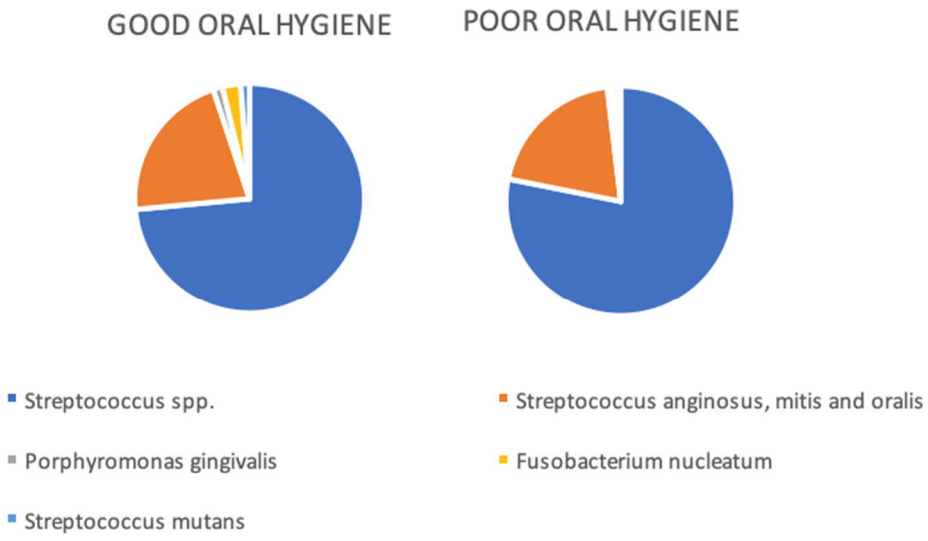


Figure captions:

Figure 1. Distribution of saliva bacteria in AT, PT and PTA patients and healthy volunteers
Figure 2. Distribution of saliva bacteria in patients with A) good oral hygiene B) poor oral hygiene